justified because of variability in an individual patient's performance with each test.

The measurements presented in our paper were indeed of the closing capacity, as Dr. Collins points out. The difference in nomenclature is unfortunate, but the manuscript was submitted to the *Journal* before the National Heart and Lung Institute document was published.

Using one standard deviation instead of two to compare our results with the predicted normal values would make no substantial difference to the conclusions, since it is the relation between the "closing volume" and functional residual capacity that is critical to gas exchange. Furthermore, the mean closing volume for the group was 125 per cent of predicted in the first two weeks after myocardial infarction, and 104 per cent of predicted ($p = 0.0008$) in the next two weeks, and every time "closing volume" was measured in the follow-up period, the value had fallen regardless of its absolute value in the immediate post-myocardial-infarction period.

Lastly, increased closing volume will lead to hypoxemia and increased alveolar-arterial oxygen gradient, if reduction in ventilation is not equally matched by a reduction in blood flow. Our paper emphasized relative hypoperfusion of the lung base, where airway closure also occurs predominantly. However, if the relative reduction in ventilation is greater than the relative reduction in perfusion, the areas of low ventilation-perfusion ($\dot{V}/\dot{Q}$) ratio in the lung will increase, and hypoxemia will ensue. This, we believe, is the predominant pathophysiologic mechanism for hypoxemia in "uncomplicated" myocardial infarction.

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CAUSES OF REVERSED PULSUS PARADOXUS

To the Editor: Massumi et al. (N Engl J Med 289:1272, 1973) recently described three causes of reversed pulsus paradoxus (inspiratory rise in the arterial blood pressure). We have observed this phenomenon in another clinical situation, during intermittent positive-pressure ventilation of a patient receiving circulatory support with an intrathoracic, intra-aortic, counterpulsation balloon pump (this balloon placed via the femoral artery and inflated during diastole boosts the cardiac output and decreases the afterload faced by the heart).

A 59-year-old patient received this therapy for circulatory failure after a ventricular aneurysmectomy and coronary-artery bypass graft. The arterial blood pressure was recorded from a radial-artery catheter via electrical transduction. The height of the peak and trough of the diastolic assist rose during inspiration, the peak rising 10 to 22 torr. A minimal increase in the systolic pressure was also noted.

It is likely that the extent of the balloon-induced blood-pressure rise is increased because the positive intrathoracic pressure (and therefore decreased intrathoracic extravascular compliance) during inspiration makes inflation of the intra-aortic balloon more effective in propelling blood. This effect is different from that noted in Massumi's patient with left ventricular failure.

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HODGKIN'S DISEASE IN SCHOOLTEACHERS (CONT.)

To the Editor: In his letter, Dr. Milham (N Engl J Med 290:1329, 1974) interpreted data from Washington State to support the hypothesis that Hodgkin's disease occurs excessively in schoolteachers. In a subsequent letter, Dr. Bahn[1] pointed out the potential bias to which Dr. Milham's method of analysis (proportional mortality) might be subject, particularly if teachers were at less risk of mortality from all causes. Indeed, there is evidence that overall mortality rates are lower for teachers than for the general population.[2]

To determine if this circumstance might obtain in the study reported by Dr. Milham, I derived the population of male teachers at risk by age in Washington State from the 1950, 1960 and 1970 censuses of the population,[4] with estimates for the intercensal years made by linear interpolation and, for 1971, by extrapolation. Average annual age-specific mortality rates from all causes and from Hodgkin's disease among all males in the state, from 1950 to 1969, were applied to these population estimates to derive the expected values in Table 1.* College professors were excluded, as I thought Dr. Milham might have done. Since data on occupation were restricted to those employed at the time of the censuses, the analyses were applied only to persons under the age of 65. Because of this restriction, the numbers of teachers, and hence the expected values, should be considered minimal estimates. The observed numbers in Table 1 are from Dr. Milham's letter.

Table 1. Observed and Expected* Deaths from All Causes and from Hodgkin's Disease among Washington State Male Schoolteachers (1950-1971).

AGE†	DEATHS FROM ALL CAUSES		DEATHS FROM HODGKIN'S DISEASE	
	OBSERVED	EXPECTED	OBSERVED	EXPECTED
20-24	16	29.09	0	0.33
25-29	43	73.94	3	1.08
30-34	35	80.12	1	1.01
35-44	98	213.32	4	1.91
45-54	220	319.09	0	1.35
55-59	132	180.84	1	0.62
60-64	174	175.32	1	0.36
Totals	718	1071.72	10	6.66
Observed/expected (relative risk)	0.7		1.5	

*Expected deaths derived by applying average-annual, age-specific rates for total Washington State male population (1950-69). †Yr.

The deficiency of deaths from all causes (relative risk = 0.7) is similar to the results of other studies.[2,3] The 10 observed deaths from Hodgkin's disease are only 3.3 more than expected (relative risk = 1.5). This excess is compatible with the reported social-class gradient for Hodgkin's disease.[5] These Washington State data suggest that the transmission of Hodgkin's disease between teachers and students is not a major epidemiologic feature of this disease.

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3. Guralnick L: Mortality by Occupation and Cause of Death among Men 20 to 64 Years of Age (Vital Statistics – Special Reports Vol 53, No 3). Washington, DC, National Center for Health Statistics, September, 1963
4. United States Bureau of the Census. US Census of Population: 1950. Vol 2, 1960: Vol 1, 1970: Vol 1, Characteristics of the Population, Washington State. Washington, DC, Government Printing Office, 1952, 1963, 1973
5. MacMahon B: Epidemiology of Hodgkin's disease. Cancer Res 26:1189-1200, 1966

*Deaths from all causes by age and sex were taken from the U. S. Vital Statistics publications for Washington State for the years 1950-69. Similar data for deaths from Hodgkin's disease were from a special tabulation provided by the National Center for Health Statistics.

CORRECT PROCEDURE FOR CEA ASSAY

To the Editor: Wu and Bray correctly state in their letter to the *Journal* that the Hoffmann-La Roche indirect assay for carcinoembryonic antigen (CEA) will not quantitate more than 25 ng per milliliter of antigen accurately (Wu JT, Bray PF: Monitoring cancer with plasma carcinoembryonic antigen. N Engl J Med 290:1439, 1974). This is clearly stated on pages 11 and 13 of the "CEA-Roche Procedure